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Equipment/Materials

A. Reflotron, Boehringer Mannheim, 9115 Hague Rd., Indianapolis, IN, 46250
B. SPSS Inc., 444 North Michigan Av., Suite 3300, Chicago, IL 60611
C. SAS Institute Inc. Box 8000, Cary, NC 27511.

Blood Lead Levels of 4–11-Year-Old Mexican American, Puerto Rican, and Cuban Children

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Synopsis

Data from the Hispanic Health and Nutrition Examination Survey were used to estimate arithmetic mean blood lead and percent with elevated blood lead [25 micrograms per deciliter (µg per dl) or greater] for 4–11-year-old Mexican American, Puerto Rican, and Cuban children. The sample size was 1,390 for Mexican American children, 397 for Puerto Rican children, and 114 for Cuban children.

Puerto Rican children had the highest mean blood lead levels (11.5 µg per dl), followed by Mexican American children (10.4 µg per dl) and Cuban children (8.6 µg per dl, P<.05*). Puerto Rican children had the highest percent with elevated blood lead (2.7 percent); 1.6 percent of Mexican American children had elevated blood lead; less than 1 percent (0.9 percent) of the Cuban children had elevated blood lead (*P<.05*). Mexican American girls had a lower mean blood lead level than did boys: 9.7 µg per dl versus 11.0 µg per dl (*P<.05*). For both Puerto Rican and Mexican American children, younger age indicated a higher risk of having elevated blood lead levels.*

*Mexican American children who lived in poverty had higher mean blood lead levels than did Mexican American children who did not live in poverty—11.6 µg per dl versus 9.6 µg per dl (*P<.05*). Despite advances in primary prevention of lead toxicity in children during the past 10 years, many Hispanic children are at risk of lead toxicity. Approximately 19,000 Mexican American 4–11-year-old children living in the Southwest and approximately 8,000 Puerto Rican children living in the New York City area had elevated blood lead levels (≥ 25 µg per dl) during 1982–84.*

AVERAGE BLOOD LEAD LEVELS of children declined nationwide from 1976 to 1980 (*1*) by approximately 37 percent. Recent advances in the primary prevention of lead toxicity in children include the phased reduction of lead in gasoline, cans of food, and paint on residential dwellings, furniture, and toys. Because leaded paint still exists in older housing units, and lead from other sources can still be found in the environment, there remains a need to monitor lead levels of children—especially children who are at high risk of lead toxicity (*2*). In a recent congressional report on the extent of lead poisoning among children, it was estimated that as many as 1.8 to 2 million children in the United States are at elevated risk for toxic lead exposure because they live in deteriorated old housing that has paint with high lead content (*3*). Earlier studies identified young children, males, blacks, those living in poverty, and those living in inner cities as being at the highest risk (*2*).

Mexican American and Puerto Rican children may also be at higher risk of lead toxicity than the general population. They have greater opportunity for exposure because of their greater likelihood of residing in inner cities and of living in poverty (*4*). Mexican American children are also more likely to use folk remedies containing high concentrations of lead (Azarcon) and to use lead-glazed pottery than the general population (*2*).

Vega-Franco and coworkers in 1978 reported a mean blood lead of 19.3 micrograms per deciliter (μg per dl) for 0–24-month-old children in Mexico (*5*). This mean blood lead level was higher than the mean of 16.3 μg per dl reported for 6–24-month-old children in the United States general population. [These data are from the second National Health and Nutrition Examination Survey (NHANES II), which was conducted during 1976–80 (*6*).] During 1970 to 1976, mean blood lead levels in New York City were found to be higher for Hispanic children (most likely Puerto Rican) than for non-Hispanic whites (*7*). Little is known about lead levels among Cuban children.

The first objective of this paper is to compare mean blood lead levels and percent with elevated blood lead for Mexican American, Puerto Rican, and Cuban children. The second objective is to examine mean blood lead and percent with elevated blood lead by the following demographic and socioeconomic indicators of exposure: age, sex, education of head of household, place of birth, central city status, and poverty.

Study Design

For this paper, the data on blood lead came from the Hispanic Health and Nutrition Examination Survey (HHANES)—a complex, multistage probability sample survey conducted during 1982 to 1984 by the National Center for Health Statistics (NCHS). The Mexican American sample consisted of children residing in households in selected counties of five southwestern States (Texas, California, Arizona, New Mexico, and Colorado). The Cuban sample was selected from the Miami-Dade County area, and the Puerto Rican sample was selected from the New York City area (parts of New York, New Jersey, and Connecticut).

The national origin or ancestry of the children was identified by either the child's parent or another adult household member during the initial interview. Mean blood lead and the percent with elevated blood lead data presented in this paper have been weighted (including noncoverage and nonresponse adjustments) to represent the noninstitutionalized population at the midpoint of data collection: Mexican American children living in the five southwestern States (March 1983), Cuban children living in Miami-Dade County (March 1984), and Puerto Rican children living in selected counties of New York, New Jersey, and Connecticut (September 1984). These point estimates represent approximately 84 percent of Mexican American, 59 percent of Puerto Rican, and 57 percent of Cuban children living in the United States during 1980. Detailed information on sample selection, coverage, and weighting process has been published (*8*).

Only lead values for 4–11-year-old children obtained by venipunctures have been included in this paper, although lead levels were assessed in 6-month- to 11-year-old children. Because of the potential for high refusal rates, blood was obtained from children ages 6 months to 3 years by fingersticks, while blood from children ages 4–11 years was obtained by venipunctures. The potential for lead contamination during large-scale fingerstick screening programs for lead has been well documented (*6*). Moreover, preliminary analysis of the NHANES II lead data suggested that including capillary blood lead data in analysis of blood lead levels would introduce bias in the estimates of mean venous blood lead levels (*5*).

Analytic Methods

Lead was measured in whole blood by atomic absorption spectroscopy, using a modification (*9*) of the Delves method (*10*), at the HANES Laboratory, Center for Environmental Health, Centers for Disease Control (CDC). The laboratory and methods used were the same as for NHANES II. Quantitation was based on the measurement of light absorbed at 283.3 nanometers (nm) by ground state atoms of lead from a hollow-cathode lamp source. Whole blood samples, human and

'This study is the first to define the subpopulations at highest risk of lead toxicity among the three largest Hispanic groups in the United States.'

bovine quality control materials, and standards (bovine whole blood spiked with aqueous lead standards) were diluted with nitric acid as the oxidizing agent, dried and ashed, and lead content was determined by using a Perkin-Elmer Model 360 or Model 2380 atomic absorption spectrophotometer with deuterium arc background correction. All materials used for collecting and processing specimens were screened for possible lead contamination, and all processing work except ashing was performed under laminar-flow hoods. Main reasons for missing lead values were refusals and insuffcent quantity for analysis. Details on venous blood sample collection, specimen storage procedures, and quality control procedures are published elsewhere (*11*).

Since the HHANES was a complex, multistage, probability sample survey, the survey design needed to be taken into account in order to estimate standard errors. For this paper, an average design effect approach was used for each of the three national origin groups. This technique has been discussed in detail in a publication (*12*).

To estimate the standard error under complex sampling, the unweighted standard error of the estimate, computed under an assumption of simple random sampling, was multiplied by the square root of the average design effect. The average design effects used for arithmetic mean blood lead were (for boys, girls, and total): 1.9, 1.9, and 2.0 for Mexican Americans; 1.3, 1.0, and 1.0 for Cubans; and 1.3, 1.2, and 1.4 for Puerto Ricans, respectively. The average design effects used for the percent of elevated blood lead were approximately 1.0. Z tests were performed using $\alpha = .05$, to test for statistical significance.

Definitions

Elevated blood lead in children was defined using the 1985 CDC recommendations of a confirmed concentration of lead in whole blood of 25 μg per dl or greater (*2*). Because of the strong relationship of blood lead of children and poverty status of the family in previous studies, the poverty index ratio was used to help identify subpopulations at risk of lead toxicity. The poverty index ratio is the annual family income adjusted for the number of persons in the family, the adult-child composition of the family, the age of the person who owned or paid the rent or mortgage for the residence (head of household), and the month and year when the family was interviewed. Children with family incomes equal to or greater than poverty thresholds had poverty indices equal to or greater than 1.0 (not poverty); those with incomes less than the poverty thresholds had indices less than 1.0 (poverty).

Results

Blood lead measurements were obtained for 1,390 Mexican American, 397 Puerto Rican, and 114 Cuban children representing final response rates of 68.6 percent of selected Mexican Americans, 59.5 percent of selected Puerto Ricans, and 49.6 percent of selected Cuban sample persons. Among examined children, those with missing blood lead values were randomly distributed by age and sex.

The arithmetic mean blood lead level was 10.4 μg per dl for Mexican American 4–11-year-olds, 11.5 μg per dl for Puerto Ricans, and 8.6 μg per dl for Cubans (table 1). These arithmetic means did not differ greatly from the geometric means: 9.4 μg per dl for Mexican Americans, 10.3 μg per dl for Puerto Ricans, and 8.2 μg per dl for Cubans. Therefore, the remaining presentation of results is retricted to arithmetic means and percent with elevated blood lead level. Although not shown, Mexican American girls had a statistically significant lower arithmetic mean blood lead level than did Mexican American boys: 9.7 μg per dl versus 11.0 μg per dl ($P<.05$). There were no significant sex differences observed among Cubans and Puerto Ricans.

The 4–11-year-olds were categorized into three groups for this analysis. As shown in table 1, mean blood lead was highest for 4–5-year-olds among Mexican Americans and Puerto Ricans ($P<.05$). The 4–5-year-olds also had the highest mean blood lead within each sex group for both Puerto Ricans and Mexican Americans (not shown). Cuban 6–8 and 9–11-year-old children had mean blood lead levels that were lower than those found for similarly aged Mexican Americans and Puerto Ricans.

As seen in table 2, 1.2 percent of the Mexican American children and 3.9 percent of the Puerto Rican children had elevated blood lead levels. Since only 1 of the 114 Cuban 4–11-year-old children (0.9 percent) had elevated blood lead, we could not continue our analysis of elevated blood lead for Cubans.

Of the 4–11-year-old Mexican American children, 4–5-year-olds had the highest percent, 4.9, with elevated blood lead ($P<.05$). For Puerto Rican children, the 4–5-year-olds had a much higher percent with elevated blood lead (10.6 percent) than did 6–8-year-olds (1.1

Table 1. Arithmetic mean blood lead in micrograms per deciliter (µg per dl) for 4–11-year-old Hispanic children by national origin or ancestry and age

Ancestry and age	Sample size	Mean level	Standard error of mean
Mexican American, total	1,390	10.4	0.19
4–5 years	269	12.2	0.55
6–8 years	542	10.4	0.28
9–11 years	579	9.5	0.26
Puerto Rican, total	397	11.5	0.34
4–5 years	67	14.5	1.12
6–8 years	165	10.9	0.42
9–11 years	165	10.8	0.52
Cuban, total	114	8.6	0.30
4–5 years	17	(1)	(1)
6–8 years	47	8.5	0.33
9–11 years	50	8.3	0.32

1 Unweighted sample size is less than 25; therefore, estimates are not given.
SOURCE: National Center for Health Statistics, HHANES, 1982–84.

Table 2. Percent of 4–11-year-old Hispanic children with elevated blood lead (25 µg per dl or more) by national origin or ancestry, sex, and age

Age and ethnic group	Sample size	Estimated population[1]	Percent elevated	Standard error
	Mexican American			
Boys, 4–11 years	707	801.6	1.6	0.5
4–5 years	150	224.0	5.6	1.9
6–8 years	277	296.5	0.3	0.3
9–11 years	280	281.1	0.7	0.5
Girls, 4–11 years	683	797.3	0.8	0.3
4–5 years	119	187.6	3.9	1.8
6–8 years	265	291.8	0.0	...
9–11 years	299	317.9	0.4	0.4
Total, 4–11 years	1,390	1,598.9	1.2	0.3
4–5 years	269	411.5	4.9	1.3
6–8 years	542	588.3	0.2	0.2
9–11 years	579	599.1	0.5	0.3
	Puerto Rican			
Boys, 4–11 years	209	103.2	2.7	1.1
4–5 years	34	24.9	[2]5.6	3.9
6–8 years	91	33.8	1.0	1.0
9–11 years	84	34.5	3.4	2.0
Girls, 4–11 years	188	102.5	5.3	1.6
4–5 years	33	24.1	[2]15.3	6.3
6–8 years	74	39.7	1.1	1.2
9–11 years	81	38.7	4.7	2.4
Total, 4–11 years	397	205.7	3.9	1.0
4–5 years	67	49.0	10.6	3.8
6–8 years	165	83.4	1.1	0.8
9–11 years	165	73.3	4.0	1.5

1 In thousands.
2 Unweighted sample size is less than 45; therefore, point estimates may not be reliable.
NOTE: Only 1 of the 114 Cuban children had elevated blood lead.
SOURCE: National Center for Health Statistics, HHANES, 1982–84.

percent), but not 9–11-year-olds ($P<.05$). Boys were the most likely to have elevated blood lead. Because only 18 (a weighted percent of 1.2) of the Mexican American 4–11-year-old children were found to have elevated blood lead, subsequent analysis has been restricted to examining mean blood lead levels.

As shown in table 3, Mexican American children who lived in poverty (poverty index ratio <1) had higher mean blood lead levels than did children who did not live in poverty: 11.6 µg per dl versus 9.6 µg per dl ($P<.05$). Mexican American children living in households whose heads had less than 7 years of schooling had the highest blood lead levels ($P<.05$). Mean blood lead for Puerto Rican children living with head of households with 0–6 years and 7–11 years of education was higher, although not statistically significant, than for Puerto Rican children living in households with the head's education of 12 years or more.

Although not shown, Mexican American children born in Mexico had higher mean blood lead levels (11.4 µg per dl) than did Mexican American children not born in Mexico (10.2 µg per dl). Children born in Puerto Rico had only slightly higher mean blood lead than did Puerto Rican children not born in Puerto Rico: 11.6 µg per dl versus 11.2 µg per dl. These differences did not reach statistical significance at the .05 level.

Discussion

This study is the first to define the subpopulations at highest risk of lead toxicity among the three largest Hispanic groups in the United States. The data described in this paper indicate that, despite advances in primary prevention of lead toxicity in children during the past 10 years, there remain many Hispanic children at risk of lead toxicity. Approximately 19,000 Mexican American 4–11-year-old children living in the southwestern United States and approximately 8,000 Puerto Rican 4–11-year-old children living in the New York City area had elevated blood lead levels ($\geq$ 25 µg per dl) during 1982–84. Lowering the cutoff for elevated blood lead to 20 µg per dl would result in an increased estimate of the number of 4–11-year-old Hispanic children with elevated blood lead: to more than 65,000 for Mexican Americans (4.1 percent), and 19,000 for Puerto Ricans (9.4 percent). Using the cutoff of 15 µg per dl raises this estimate even further: to 240,000 (15 percent) Mexican Americans and 41,000 (20.1 percent) Puerto Ricans.

Approximately 1 of 9 Puerto Rican and 1 of 20 Mexican American 4–5-year-old children were found to have elevated blood lead levels greater than 24 µg per dl during 1982–84. A reanalysis of the NHANES II lead data for 4–5-year-olds showed that approximately 1 of 29 non-Hispanic white 4–5-year-olds and 1 of 5 non-Hispanic black 4–5-year-olds had elevated blood

Table 3. Arithmetic mean blood lead in µg per dl for 4–11-year-old Hispanic children by national origin or ancestry and household descriptors

	Mexican American			Puerto Rican		
Category	Sample size	Mean blood lead level	Standard error of mean	Sample size	Mean blood lead level	Standard error of mean
Poverty index ratio:						
Poverty (<1)	513	11.6	0.33	237	11.9	0.41
Not poverty (≥ 1)	773	9.6	0.24	137	11.0	0.65
Education of head of household:						
0–6 years	473	12.0	0.39	58	11.7	1.02
7–11 years	378	10.1	0.29	183	12.0	0.53
High school or more	508	9.2	0.27	143	10.7	0.47

SOURCE: National Center for Health Statistics, HHANES, 1982–84.

lead in 1976–80 (*5*). A comparison of these ratios suggests that, even though mean blood lead levels declined during the late 1970s, both Mexican American and Puerto Rican prevalence rates of elevated blood lead were higher during 1982–84 than non-Hispanic white rates were for 1976–80, but not as high as for non-Hispanic blacks.

In NHANES II, boys 6 months to 5 years had a nonstatistically significant higher mean blood lead and percent with elevated blood lead than girls (*5*). Among 6–17-year-olds, the difference in blood lead levels between boys and girls increased with age, with boys having higher mean blood lead and percent elevated blood lead than girls (*13*). Mexican Americans showed the same sex differences that were found in NHANES II: Mexican American boys had a statistically significant higher mean blood lead and a nonstatistically significant higher percent elevated blood lead than did Mexican American girls.

When making comparisons among subpopulations such as we have made in this paper, it is important to keep in mind that any differences in mean blood lead and the percent with elevated blood lead that were found may reflect differences in lead exposure, lead absorption, or the metabolic response to lead. No information on possible environmental sources of lead, such as the presence of peeling lead-based paint in the residence, was gathered in HHANES.

Lead-based paint, or soil or dust contaminated with it, is considered to be a major source of high blood lead levels in children (*2*). Children ingest lead by eating paint chips or chewing objects such as cribs that are coated with lead-based paint. The youngest children are at greatest risk of ingesting lead-based paint chips since they are more prone to exhibit pica and normal mouthing behavior. Previous studies have found that children living in dilapidated old housing (usually low-income children living in central cities) are most likely to come into contact with peeling lead-based paint (*2*). For both Mexican Americans and Puerto Ricans, 4–5-year-olds had the highest percent elevated blood lead and mean blood lead levels of all 4–11-year-old children. Although not presented in this paper, consistent with NHANES II findings, children living in central cities had the highest mean blood lead among both Mexican American and Puerto Rican 4–11-year-old children. These differences did not reach statistical significance.

Mean blood lead levels were examined by birthplace to identify possible sources of lead exposure. No information was collected, though, on how long the person had lived in the United States, whether there has been continued transmigration between the mainland United States and Puerto Rico or Mexico (especially relevant for Puerto Ricans), and exactly where in each country the person in the sample had lived.

This study agrees with previous findings that Puerto Rican children living in the New York City area have a higher risk of lead toxicity than most other subpopulations. This may be due to the high proportion (42 percent) of Puerto Ricans who live in poverty (*14*)—a risk factor for living in deteriorated housing with peeling lead-based paint. Mexican American children living in the southwest United States may share some of this higher risk since 23 percent of Mexican Americans, twice the 11 percent for non-Hispanics, live in poverty. In comparison, 36 percent of blacks live in poverty (*14*). Screening programs to identify children with elevated blood lead levels should continue to focus their efforts toward younger children among Puerto Ricans living in the New York City area and younger children among Mexican Americans living in poverty in the southwestern United States.

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Mortality from Diabetes Mellitus, Ischemic Heart Disease, and Cerebrovascular Disease Among Blacks in a Higher Income Area

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Synopsis ....

According to the 1980 census, blacks in Suffolk County on Long Island, NY, had a median family income of almost $20,000 versus $12,618 for blacks in the entire United States, or only 20 percent lower than that for whites in the county. Black-white ratios of age-specific death rates for 1979–83 in Suffolk County were elevated for all causes for men and women in age groups from 35–44 to 55–64 years (but not for those 75 years or older), for ischemic heart disease for women (but not men) for age groups from 35–44 to 55–64 years, for diabetes mellitus for most ages (especially for females), and for cerebrovascular disease for both men and women for all age groups from 35–44 to 65–74 years.

The age-specific proportional mortality ratios (PMRs) for ischemic heart disease within educational level (less than 12 years and 12 or more years of school) were lower for black than for white men but more similar for black and white women. For diabetes, the PMRs were higher for black versus white women within both educational levels. PMRs for cerebrovascular disease were higher for black than white men within the group of decedents with less than 12 years of education. The findings are discussed with reference to racial differences in the prevalence of poverty as well as possible differences in risk factors (for example, obesity) or medical care independent of poverty.

STUDIES HAVE REPORTED black-white differences in mortality in economically depressed areas in Chicago (*1*), Alameda County (*2*), and Newark (*3*). Differences in socioeconomic status (SES) and poverty level are major explanations for black-white differences in total mortality (*4*) and in mortality for certain causes of death. An ecologic study in Philadelphia (*5*), however, showed that higher death rates in geographic areas containing low-income groups or higher proportions of blacks did not hold for ischemic heart disease for both sexes or for diabetes mellitus for males.

There is increasing interest in geographic differences